Epidemiological data such as ours are vulnerable to many sources of bias. Outpatient facilities at health centres improved during the study, but this should have had only a minor effect on the rate of attack in children under 1 and no effect on the prevalence of free treatment. The ratio of prevalence of free treatment to rate of attack remained constant at 2·9 in 1978 and 2·8 in 1984, suggesting that the criteria for admission to hospital did not change during the study. The data from all three of our sources consistently showed an appreciable decrease in the rates of infection.

We could not collect any data on possible factors leading to the decline in urinary tract infection. One important possible factor is the simultaneous improvement in the diagnosis of such infections: their diagnosis, treatment, and consequences have been subjects of postgraduate courses for both general practitioners and paediatricians. In the light of our results showing a continuous decline in the treatment of urinary tract infections, both in hospitals and by continuous treatment, a hypothesis of an improvement in diagnosis suggests that medical practice has been unsatisfactory. The decrease we observed in the number of urine samples giving positive results on bacterial culture is, however, difficult to explain in terms of improvements in diagnosis.

It is tempting to connect our findings with the simultaneous changes in the care of infants. In particular, the use of napkins changed appreciably during the study, disposable napkins of better quality being used increasingly. The impression of paediatricians working in developing countries where napkins are not used is that the rate of urinary tract infections is low.[3] A child who has had one symptomatic infection runs an increased risk of having another for a long time afterwards.[9] Thus factors affecting the disease in early infancy could be reflected in its occurrence even in later childhood. We suggest that the decrease in symptomatic urinary tract infections shown by our results may be associated with changes in caring for infants.

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1 Wettegren B, Jodal U, Jonasson G. Epidemiology of bacteriuria during the first year of life. *Acta Paediatr Scand* 1985;**74**:925-33.
2 Roberts JA. Urinary tract infections. *Am J Kidney Dis* 1984;**4**:103-17.
3 Smallpeice V. *Urinary tract infection in childhood and its relevance to disease in adult life*. London: William Heinemann, 1968:1-65.
4 Stansfeld JM. Clinical observations relating to incidence and aetiology of urinary tract infections in children. *Br Med J* 1966;i:631-5.
5 Winberg J, Andersen HJ, Bergstrom T, Jacobsson B, Larson H, Lincoln K. Epidemiology of symptomatic urinary tract infection in childhood. *Acta Paediatr Scand* [Suppl]1974;**252**:1-20.
6 Last JM. *A dictionary of epidemiology*. New York: Oxford University Press, 1983:82.
7 Bailar JC. Significance factors for true ratio of a Poisson variable to its expectation. *Biometrics* 1964;**20**:639-43.
8 Wiswell TE, Roscelli JD. Corroborative evidence for the decreased incidence of urinary tract infections in circumcised male infants. *Pediatrics* 1986;**78**:96-9.
9 Smellie JM, Hodson CJ, Edwards D, Normand ICS. Clinical and radiological features of urinary infections in childhood. *Br Med J* 1964;ii:1222-6.

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Painful crises in sickle cell disease—patients' perspectives

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Abstract

One hundred and two patients returned structured questionnaires sent to clinics for sickle cell disease in the United Kingdom in order to gain greater insight into the patients' perception of painful crises. Most patients who suffer pain crises experience a prodromal stage that should be investigated further to find out if prophylaxis is possible. Cold, exertion, and tiredness were the most important precipitating factors. Despite the increase in the amount of knowledge about sickle cell disease in recent years, and though 29 out of 88 patients (30-40%) believed that medical services were improving, 33 out of 88 (30-40%) were still experiencing long delays in being treated in hospital. A third of patients do not seem to receive adequate pain relief.

Introduction

Sickle cell disease is common in urban areas; in London alone there are nearly 2000 patients.[1] Painful vaso-occlusive crises account for nearly all the acute hospital admissions of patients with sickle cell disease in the London area.[2] These episodes vary in severity and duration within the same patient as well as between individual patients.[3] The physical signs and symptoms have been extensively reviewed but much remains to be learned about the patients' own perceptions of their illnesses. This paper describes an attempt to collect information from patients on various aspects of management of pain crises both at home and in hospital.

Patients and methods

A structured questionnaire was used to obtain information about age, sex, the patients' awareness of incipient sickling crises and measures taken to avert or control them, previous sources of health education, the accessibility and adequacy of medical care, and perception of factors causing crises and measures taken to reduce the pain.

Information about the intensity of pain during the patient's last crisis, about the problems of the doctors' and nurses' understanding of the amount of pain, and about the problems of sickle cell disease, were obtained with a 0-5 linear analogue scale. A 0-3 linear analogue scale was used to measure patients' perception of pain relief. Other questions were answered by ticking the appropriate box, and space was left for additional comments. More than one answer to particular questions could be given.

The data were collected over the three months June to September 1986 from patients attending sickle cell and haematology clinics in London, Birmingham, and Manchester. The diagnostic criteria have been summarised by Serjeant.[4] Four hundred questionnaires were sent to the clinics for distribution, and 102 patients (61 women and 41 men) aged between 11 and 49 years returned the questionnaire. The response rate is unknown because the number of questionnaires given to patients is unknown. Of the responders, 77 were homozygous for haemoglobin S, 21 were doubly heterozygous for haemoglobin S and haemoglobin C, one had haemoglobin S-β thalassaemia, one was doubly heterozygous for haemoglobin S and haemoglobin J, and two had sickle cell disease but were uncertain of their genotypes. Three of those homozygous for haemoglobin S, five who were homozygous for haemoglobin S and haemoglobin C, and both those of unknown genotype, had never had a painful crisis. A further four patients who were homozygous for haemoglobin S had not been admitted to hospital because of a painful crisis.

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TABLE I—*Duration of prodromal phases. Several respondents gave more than one time (n=59)*

Duration of prodromal phases	No (%) of patients
<1 Hour	59 (100)
1-12 Hours	29 (49)
12-24 Hours	19 (32)
"Days"	15 (26)
"Weeks"	3 (5)

Results

The Mann-Whitney test for non-parametric, independent proportions showed no significant differences between groups and so the results were combined.

Prodromal stage—Fifty nine patients reported that they had a premonition of a painful crisis. Symptoms were imprecise, but included feelings of numbness, aches, pins and needles in the areas affected later, or just an awareness that pain would follow. The interval between prodrome and onset of pain varied among patients and for the same patient (table I).

Personal management of pain was similar in the periods before and during periods of pain. Keeping warm, taking extra fluids, rest, and taking painkilling drugs were the main forms of self treatment. Relaxation (31 out of 58) was more commonly practised in the period before pain than during the pain (25 out of 92). Taking extra vitamins (nine out of 92) and herbal remedies (nine out of 92) and talking about fears or feelings (eight out of 92) were done less often during the painful crises.

Measures to avoid painful crisis—Factors known to precipitate a crisis were cold (n=67), exertion or tiredness (n=60), infection (n=48), stress or worry (n=44), dehydration (n=32), alcohol consumption (n=15), and pregnancy (n=9). Measures taken to try to avoid crises were drinking plenty of fluids (n=75), keeping warm (n=55), avoiding stress (n=29), taking vitamins (n=25), and avoiding tiredness (n=6).

Eighty one respondents said that they had been given some advice on self management of sickle cell disease. The hospital doctor was the main source of information (n=45), followed by family and friends (n=30), the local sickle cell centre (n=16), the Sickle Cell Society (n=14), the Organisation for Sickle Cell Anaemia Research (n=6), and the general practitioner (n=5). Of those who had received information from any source, 79 thought that it was comprehensible and 70 of these believed that it had helped them.

Pain management at hospital—Those patients who could not manage their own painful crises presented at accident and emergency departments, or were referred by their general practitioners to haematology departments (n=88). Most patients felt that the general practitioners and the staff in the accident and emergency departments were the least able to understand the problems of sickle cell disease, whereas staff on the hospital ward seemed to show greater understanding (table II). This seems to be partly associated with the effectiveness of the painkilling drugs that were prescribed (table III).

TABLE II—*Patients' measurements on 0-5 linear analogue scale of degree of understanding shown by general practitioners and hospital staff (n=88)*

	Scores allotted by patients				
	0-1	1-2	2-3	3-4	4-5
General practitioners	28	13	18	10	19
Staff in accident and emergency departments	43	13	14	7	11
Staff in hospital wards	15	6	16	19	32

Of the 88 patients who sought professional help at the hospital, 18 thought that they were seen quickly, and 33 thought that the delay was too long. Comparing present treatment with previous experience, 53 thought that doctors and nurses were more knowledgeable about sickle cell disease and 54 thought that the pain killing drugs they were given were more effective. Only one third, however, thought that they were now treated more quickly, and only 34 thought that once they had been seen pain relief was given more quickly.

Of the 92 patients using painkilling drugs for the painful crisis, 40 were worried about the drug they were given. Where specified their concerns were side effects (n=17), overdosage (n=1), and addiction (n=6). In hospital only 23 routinely received their analgesics on demand. When asked whether pain relief was there when needed 40 said that it was. Twenty nine thought that pain relief was not always given when needed.

TABLE III—*Association between degree of understanding shown by general practitioners and hospital staff, and amount of pain relief received. Figures are given as number (percentage)*

	Degree of understanding*			
	General practitioners		Hospital staff	
	0-2·4	2·5-5·0	0-2·4	2·5-5·0
Amount of pain relief:†				
0-1	41 (47)	20 (23)	9 (10)	5 (6)
2-3	8 (9)	19 (21)	20 (23)	54 (61)

*Measured on a linear analogue scale 0-5.
†Measured on a linear analogue scale 0-3.

When patients assessed their pain on a 0-5 linear analogue scale the mean score was 4·42, with a skewed distribution of mode 5 and median 5. Patients assessed the doctors' and nurses' appreciation of the same pain as having only a mean of 2·26 with a mode and median of 2. Fifty seven patients thought that staff did not appreciate the amount of pain they were having.

Discussion

The results are biased in that the responders were all regular attenders of outpatient clinics and sickle cell centres, and therefore represent those patients with sickle cell disease who are dependent on professional support. Furthermore, they were drawn from those haematology departments and sickle cell centres that were interested enough to cooperate with this study, and they may therefore be receiving the best care available.

Cold and tiredness were the most likely factors to precipitate crises. In a recent report by Black and Laws from Newham (with a smaller number of patients) the results were similar.[5] It is important that these factors are appreciated by doctors, nurses, and social workers. Patients should be as strongly advised to keep warm and avoid tiredness as to drink plenty of fluids, and should be given whatever help is available to achieve these ends.

Education of both patients and their families about how to avoid crises may lead to a decrease in their number and severity. In spite of this group of patients being biased in favour of those attending hospitals for treatment, over 30% had either not received advice (22%), or felt that the advice given was of no benefit. Hospital staff may not appreciate fully that they are the usual source of such advice, but they may not have the knowledge, training, or the time to give it. Counsellors from sickle cell centres could be present at outpatient clinics (as occurs in some hospitals), to help with both clinical and social problems, or patients could be referred to a counsellor based elsewhere.

A prodromal stage was common and usually short, though sometimes up to 24 hours elapsed before the painful crisis occurred. This time could be exploited to start prophylaxis, because keeping pain at bay with analgesics is easier than stopping it once it has started. Furthermore, lower doses of analgesics are usually required. As nearly half the patients were anxious about the painkilling drugs that they were given, this would clearly be of benefit.

Drug addiction and dependence are potentially

serious problems, but they are sometimes mentioned in an alarmist and unhelpful way by members of staff.[6] Patients' main worries were about the side effects of the drugs rather than about addiction, and so optimal management of pain should include adequate reassurance about these fears.

There were significant differences in the degrees of pain perceived by patients and those that they thought the staff perceived. For some patients pain relief was inadequate, and not always given swiftly enough. There seems to be a need for more formal monitoring of pain relief by nursing and medical staff. Also, because pain comes and goes during a crisis, patients should have some control over the amount of analgesia they are given. One can treat different degrees of pain using a simple visual analogue scale and achieve both these objectives.[7]

In conclusion, this study highlights the need for reappraisals of the management of pain in sickle cell disease, and of how pain is monitored and treated. The use of counsellors to help in advising patients on the avoidance of the crises should be encouraged. A prodromal stage of the painful crisis was common, and this could be used to initiate early treatment.

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1 Anionwu E, Walford D, Brozovic M, *et al.* Sickle cell disease in a British urban community. *Br Med J* 1981;**282**:283-6.
2 Brozovic M, Anionwu E. Sickle cell disease in Britain. *J Clin Pathol* 1984;**37**:1321-6.
3 Diggs LW. Sickle cell crisis. *Am J Clin Pathol* 1965;**44**:1-19.
4 Serjeant GR. *Sickle cell disease*. Oxford: Oxford University Press, 1985:34-47.
5 Black J, Laws S. *Living with sickle cell disease*. London: Sickle Cell Society, 1986:43.
6 Sickle Cell Society. *Pain in sickle cell disease*. London: National Extension College, 1985:7.
7 Revill SI, Robinson JO, Rosen M, *et al.* Reliability of a linear analogue for evaluating pain. *Anaesthesia* 1976;**31**:1191-6.

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Effect of dietary salt on bronchial reactivity to histamine in asthma

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In a recent epidemiological survey increased dietary salt intake was positively associated with bronchial reactivity to inhaled histamine.[1] If this association is important in the pathogenesis of asthma a similar relation would be expected in individual patients. We therefore investigated the effect of increasing salt intake on bronchial activity in a group of asthmatic patients and healthy non-asthmatic volunteers.

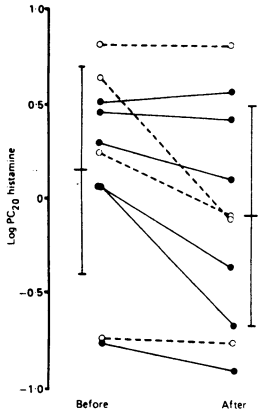
Patients, methods, and results

Ten asthmatic patients who attended the hospital outpatient department were studied (six men, four women; age range 18-63 years). Five patients were atopic and five non-atopic. All had a history of intermittent wheezing and a greater than 15% change in forced expiratory volume in one second either spontaneously or after treatment. Three were taking an inhaled β_2 agonist daily, and seven were also receiving an inhaled steroid. All were non-smokers. Five non-asthmatic subjects (one man, four women; age range 23-48 years) volunteered to act as controls.

The dietary salt intake of all participants was assessed by a dietitian. Salt excretion was measured in two 24 hour urinary specimens, and the average was recorded. A histamine challenge test was performed with a modification of that described by Cockcroft *et al.*[2] Doubling doses of histamine in phosphate buffered saline were inhaled, from 0·09 mmol/l to 26·04 mmol/l. The forced expiratory volume in one second was measured one and two minutes after each inhalation, and the test was stopped when the volume decreased by 20% or more. The histamine concentration that produced a decrease in forced expiratory volume of 20% (PC_{20}) was calculated by linear interpolation of the last two points on the log dose response curve. All challenge tests were performed at midday, bronchodilator treatment having been withdrawn 12 hours beforehand.

Salt intake was then doubled in all participants by increasing dietary salt and also by giving them salt tablets. At the end of one month the histamine challenge was repeated. For each participant the repeat baseline forced expiratory volume in one second was within 0·25 litres of the value recorded at the initial test. Measurement of 24 hour urinary salt excretion was also performed at that time.

The results were compared by Student's paired *t* test. For the histamine results this was done after logarithmic transformation. There was a significant increase in bronchial reactivity to histamine in nine of the 10 asthmatic patients when their salt intake was increased. The PC_{20} decreased from a mean (SD) of 7·49 (6·51) mmol/l to 5·2 (6·51) mmol/l ($p<0·05$) (figure). Reactivity in the controls was normal and did not change on either occasion. The 24 hour salt excretion increased from 156 (50) mmol/24 h to 216 (39) mmol/24 h ($p<0·0005$) in the asthmatics and from 103 (22) mmol/24 h to 183 (48) mmol/24 h ($p<0·05$) in the controls.



Logarithm of histamine concentration that produced 20% decrease (PC_{20}) in forced expiratory volume in one second before and after increased salt intake in asthmatic patients. ●=Men. ○=Women. Bars indicate mean and SD.

Comment

Another British study has shown that increased salt intake increases bronchial reactivity.[3] It suggested, however, that this phenomenon was restricted only to men, whereas we have shown this to occur in women asthmatics as well but not in healthy control subjects.

The way in which increased salt intake may cause increased bronchial hyperreactivity is unclear. Animal studies have shown that sensitised smooth muscle shows hyperreactivity as a consequence of increased activity of the cell membrane sodium pump and sodium influx.[4] Increased sodium intake may possibly augment this effect.[1] As the degree of histamine reactivity is related to the severity of symptoms in asthma[5] these results also indicate that a high dietary salt intake may contribute to the severity of asthma.

Further studies are needed to gauge the importance of salt intake in determining this severity and especially the role that salt restriction may have in its treatment.

1 Burney PGJ, Britton JR, Chinn S, *et al.* Response to inhaled histamine and 24 hour sodium excretion. *Br Med J* 1986;**292**:1483-6.
2 Cockcroft DW, Killian DN, Mellon JJA, Hargreave FE. Bronchial reactivity to inhaled histamine: a method and clincal survey. *Clin Allergy* 1977;7:235-43.
3 Burney PGT, Neild J, Twort C, *et al.* The effect of changing dietary sodium on the bronchial response to histamine. *Thorax* (in press).
4 Souhrada M, Souhrada JF. Sensitization induced sodium reflex in airway smooth muscle cells. *Am Rev Respir Dis* 1985;**131**:A356.
5 Juniper EF, Frith PA, Hargreaves FE. Airways responsiveness to histamine and metacholine: relationship to minimum treatment to control symptoms of asthma. *Thorax* 1981;**36**:575-9.

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